Acute Respiratory Failure in Severe Acute Brain Injury

Zachary Robateau, MD[a,*], Victor Lin, MD[a],
Sarah Wahlster, MD FNCS[a,b,c]

KEYWORDS

• Severe acute brain injury • Traumatic brain injury • Acute ischemic stroke
• Intracranial hemorrhage • Subarachnoid hemorrhage • Cardiac arrest
• Acute respiratory failure • Acute respiratory distress syndrome

KEY POINTS

- Patients with severe acute brain injury have been excluded in most studies guiding mechanical ventilation strategies or acute respiratory distress syndrome management, and there is limited evidence to inform ventilatory strategies in this population.
- Cerebral and pulmonary pathophysiology are intricately intertwined via complex, often bidirectional pathways involving elevated intracranial pressure (ICP), systemic inflammatory pathways and neuroinflammation, hormonal dysregulation, and catecholamine surges.
- Brain-injured patients likely require higher arterial oxygen partial pressure targets than other critically ill patients; however, hyperoxemia is also detrimental. Effects on the cerebral vasculature, ICP, and cerebral perfusion should be considered when determining arterial carbon dioxide partial pressure targets.
- Vigilant monitoring is warranted in patients at risk for elevated ICP with low tidal volume ventilation utilization, positive end-expiratory pressure titration, optimization of plateau pressure and driving pressure, and prone positioning.
- The ability to predict successful extubation in brain-injured patients remains a challenge. There is no benefit of early (<7 days) tracheostomy in stroke, more data are needed in traumatic brain injury.

INTRODUCTION

Severe acute brain injury (SABI) accounts for up to 25% of individuals receiving mechanical ventilation (MV).[1,2] Yet, there are insufficient data to inform ventilatory targets in this population because patients with SABI, in particular those with elevated

[a] Department of Neurology, University of Washington, Seattle, USA; [b] Department of Neurological Surgery, University of Washington, Seattle, USA; [c] Department of Anesthesiology and Pain Medicine, University of Washington, Seattle, USA
* Corresponding author.
E-mail address: zrobateau@gmail.com

Crit Care Clin ■ (2024) ■–■
https://doi.org/10.1016/j.ccc.2024.01.006 **criticalcare.theclinics.com**
0749-0704/24/© 2024 Elsevier Inc. All rights reserved.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

SHOUT
SHARE
IT

intracranial pressure (ICP), have frequently been excluded in landmark trials evaluating MV strategies.[3–6]

SABI refers to sudden onset traumatic or nontraumatic injury to the brain that results in decreased level of consciousness, often in combination with other substantial neurologic deficits. The term SABI encompasses several distinct pathologic conditions, including acute ischemic stroke (AIS), intracerebral hemorrhage (ICH), subarachnoid hemorrhage (SAH), traumatic brain injury (TBI), and hypoxemic-ischemic encephalopathy after cardiac arrest. Current management is focused on minimizing secondary brain injury by optimizing cerebral perfusion and oxygen supply while accounting for ICP, cerebral perfusion pressure (CPP), cerebral oxygen demand and delivery, seizures, and systemic hemodynamics. The long-term neurologic prognosis is often difficult to predict in the acute stage and critical, time-sensitive decisions regarding invasive treatments versus withdrawal of life-sustaining treatment must be made in the face of this prognostic uncertainty.

There are some unique, clinically relevant considerations in managing acute respiratory failure in SABI: (1) Cerebral and pulmonary pathophysiology are intricately intertwined via complex, often bidirectional pathways; (2) The optimal arterial blood gas targets may be different in patients with SABI to optimize ICP, cerebral perfusion, or cerebral oxygen delivery; (3) Lung-protective strategies established in the general critical care population may be harmful in SABI and conflict with neuroprotective measures, especially in patients with elevated ICP; and (4) There are substantial uncertainties around ventilator liberation strategies, gauging adequate airway protection, and predicting the ability to extubate patients with SABI.

The goal of this article is to summarize the relevant evidence in this population, highlight commonly encountered clinical challenges, specifically brain–lung conflict in concomitant SABI and acute respiratory distress syndrome (ARDS), and provide guidance by reviewing important practical considerations in providing care to critically ill brain-injured patients who require MV.

BRAIN–LUNG INTERACTIONS

The interplay between the brain and lungs is intricate, dynamic, and often bidirectional.[7,8] Although not yet fully understood, pathophysiological interactions are thought to be mediated via elevated ICP, neuroinflammatory and systemic inflammatory responses, hormonal dysregulation, catecholamine surges, and dysregulation of central breathing control (**Fig. 1**). Additional factors commonly encountered in critically ill patients, such as hypotension and shock, sedation and polypharmacy, fever, and delirium, may additionally affect these interactions.

Hypoxemia and inflammation due to acute lung injury can worsen secondary brain injury. ARDS alone has been linked to substantial long-term neurocognitive impairment through various mechanisms, such as acute blood brain barrier (BBB) disruption, altered cerebral blood flow, and hippocampal neuronal damage.[9–11] Conversely, SABI may precipitate secondary lung injury through various mechanisms (see **Fig. 1**), including neurogenic pulmonary edema (NPE).[8,12] SABI can also induce or worsen ARDS and has been reported in 4% to 48% of patients with SABI.[7,11,13–22]

Clinically, it is challenging to distinguish NPE from ARDS, and there is substantial overlap in pathophysiology between the 2 entities. Current theories suggest distinct underlying mechanisms: NPE is thought to be a consequence of excessive sympathetic activity, resulting in alterations in pulmonary vascular resistance and capillary permeability ultimately causing volume overload, whereas ARDS is mediated by inflammation, resulting in direct intrapulmonary tissue injury.[7,8,12] However, histopathological

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

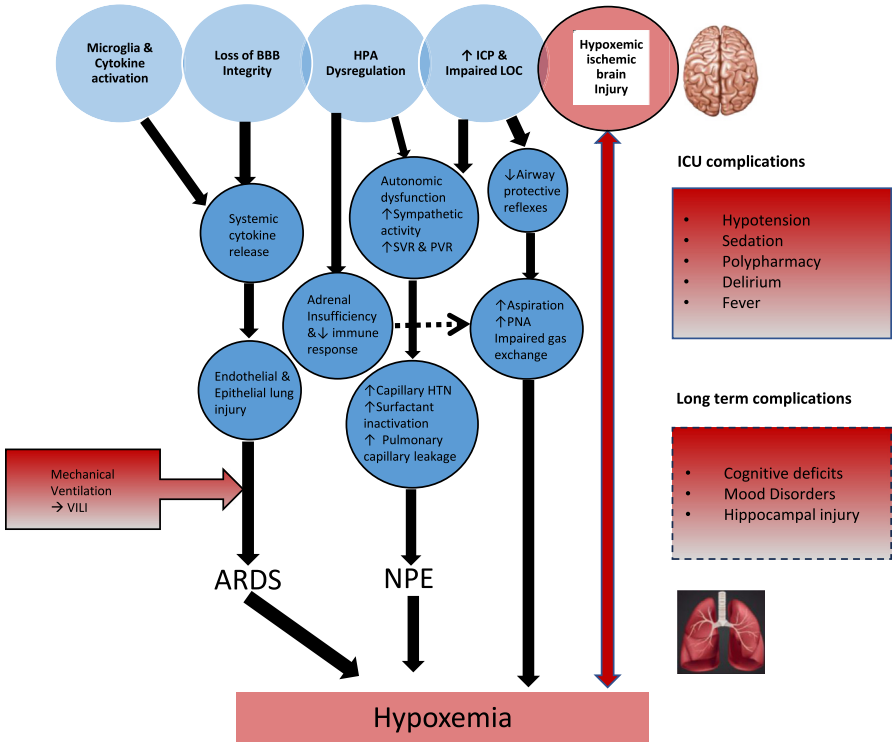

Fig. 1. Brain–lung interactions. ARDS, acute respiratory distress syndrome; HPA, hypothalamic-pituitary-adrenal; HTN, hypertension; ICP, intracranial pressure; ICU, intensive care unit; LOC, level of consciousness; NPE, neurogenic pulmonary edema; PNA, pneumonia; PVR, peripheral vascular resistance; SVR, systemic vascular resistance; VILI, ventilator-induced lung injury.

postmortem analyses suggest similar features, and there are likely bidirectional interactions, and ultimately, clinical management is similar.

INDICATIONS FOR MECHANICAL VENTILATION IN SEVERE ACUTE BRAIN INJURY

Patients with SABI commonly require intubation due to insufficient airway protection in the context of decreased level of consciousness, at times coupled with impaired respiratory drive or cranial nerve dysfunction. Other frequently encountered indications for intubation in SABI include treatment of seizures or status epilepticus, ICP management, to facilitate urgent neuroimaging or procedures, or extreme agitation. Patients with SABI are also at high risk for secondary pulmonary complications including aspiration and/or pneumonia, pulmonary embolism, pulmonary edema, as well as pulmonary contusions or hemothorax/pneumothorax in the context of trauma.

The widely established mantra that a patient with glasgow coma scale (GCS) less than 8 requires immediate intubation was primarily established in trauma populations and is often reflected in published guidelines; however, this practice has since been challenged and may even be harmful in some scenarios, with recent studies suggesting poor outcomes.[23–26] GCS may also be confounded by various sequelae of SABI, such as the presence of aphasia or eyelid opening apraxia, and therefore needs to be interpreted with consideration of affected neuroanatomical structures. When assessing airway protection and the need for intubation, objective signs to suggest compromise

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

of respiratory function include visual observation for evidence of airway obstruction (jaw and tongue prolapsing posteriorly, retractions, and paradoxic thoraco-abdominal movement) or upper airway noises (snoring, stridor, stertor, and gurgling of oropharyngeal secretions). An inadequate cough is another concerning sign, especially in combination with pooling secretions and need for frequent suctioning; the role of the gag reflex is less clear. Ultimately, changes in oxygenation and ventilation should be closely monitored and guide decisions regarding intubation. Hypoxia is widely recognized to result in worsened outcomes in SABI, presumably due to secondary cerebral injury from cerebral metabolic demand mismatch with low oxygen delivery to the hypermetabolic, acutely injured brain.[27–30] Ineffective or insufficient ventilation can result in hypercarbia, which is a potent mediator of cerebral vasodilation and can cause or worsen elevated ICP. In contrast, abnormal respiratory patterns such as central hyperventilation can cause hypocapnia and cerebral vasoconstriction, which may subsequently precipitate cerebral ischemia.[31–33]

The optimal intubation strategy for patients with SABI depends on individual patient characteristics, including concerns about elevated ICP, impaired cerebral autoregulation, insufficient cerebral perfusion (especially in patients with cerebrovascular stenoses, occlusions, dissections, or vasospasm), cervical spine stability in patients with trauma, and presence of concomitant heart failure. Rapid sequence intubation has been reported to be successful in 90%; however, complications have been described in 20% to 25%—most commonly hypotension and hypoxemia—with life-threating complications in 2%.[13] Unique considerations to minimize secondary brain injury in SABI include induction techniques aiming for hemodynamic stability to avoid systemic blood pressure fluctuations, including rapid drops (especially in patients with impaired perfusion) and spikes (especially in patients with ruptured vascular lesions or acute ICH), choosing class and dose of neuromuscular blocking agent, and deliberate utilization of end tidal CO_2 monitoring.[34]

In SABI, propofol is a commonly chosen induction agent because it can blunt cerebral metabolism and therefore decrease cerebral blood flow demand but the clinician must be proactively aware and prepared to treat hypotension. Lidocaine can be used to protect against transient ICP elevations by blunting the sympathetic response, although data supporting its use is limited.[35] Similarly, the use of a nondepolarizing neuromuscular blocking agent can obviate or blunt the cough reflex and minimize ventilator dyssynchrony (VD) for the first ~30 to 90 minutes after administration.

VENTILATOR MANAGEMENT

A recent consensus guideline by the European Society of Intensive Care Medicine (ESICM) emphasizes the lack of evidence to guide ventilator management in SABI, specifically in patients with increased ICP and ARDS.[36] The following sections review the most recent updates and relevant considerations for various ventilator parameters in this population (**Fig. 2**).

Ventilator Mode

To date, it has not been established whether there is an optimal mode of ventilation in SABI, with no major trials directly comparing strategies primarily using controlled versus spontaneous modes. A controlled mode is beneficial to achieve specific arterial carbon dioxide partial pressure ($Paco_2$) and pH targets in the context of elevated ICP. Spontaneous modes can be used in patients with minimal oxygen requirements and intact respiratory drive, and may mimic a more physiologic state, thus increasing patient comfort and requiring decreased levels of sedation. Controlled modes may also exacerbate

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

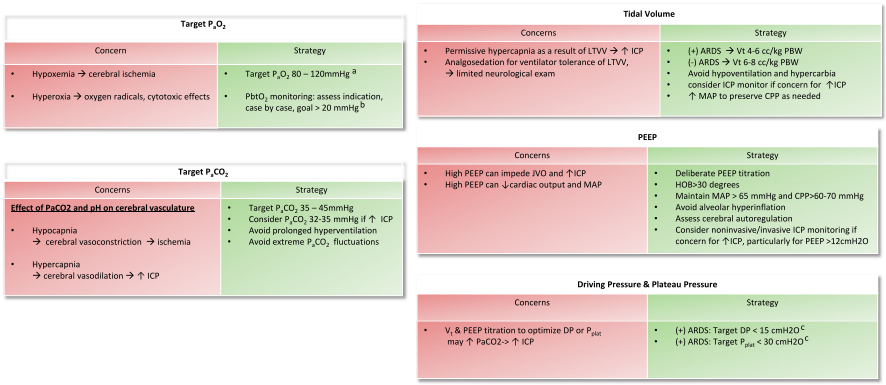
Target P_aO_2
Concern
Strategy
Hypoxemia → cerebral ischemia
Hyperoxia → oxygen radicals, cytotoxic effects
Target P_aO_2 80 – 120mmHg [a]
$PbtO_2$ monitoring: assess indication, case by case, goal > 20 mmHg [b]
Target P_aCO_2
Concerns
Strategy
Effect of PaCO2 and pH on cerebral vasculature
Hypocapnia → cerebral vasoconstriction → ischemia
Hypercapnia → cerebral vasodilation → ↑ ICP
Target P_aCO_2 35 – 45mmHg
Consider P_aCO_2 32-35 mmHg if ↑ ICP
Avoid prolonged hyperventilation
Avoid extreme P_aCO_2 fluctuations
Tidal Volume
Concerns
Strategy
Permissive hypercapnia as a result of LTVV → ↑ ICP
Analgosedation for ventilator tolerance of LTVV, → limited neurological exam
(+) ARDS → Vt 4-6 cc/kg PBW
(-) ARDS → Vt 6-8 cc/kg PBW
Avoid hypoventilation and hypercarbia
consider ICP monitor if concern for ↑ICP
↑ MAP to preserve CPP as needed
PEEP
Concerns
Strategy
High PEEP can impede JVO and ↑ICP
High PEEP can ↓cardiac output and MAP
Deliberate PEEP titration
HOB>30 degrees
Maintain MAP > 65 mmHg and CPP>60-70 mmHg
Avoid alveolar hyperinflation
Assess cerebral autoregulation
Consider noninvasive/invasive ICP monitoring if concern for ↑ICP, particularly for PEEP >12cmH2O
Driving Pressure & Plateau Pressure
Concerns
Strategy
V_t & PEEP titration to optimize DP or P_{plat} may ↑ PaCO2-> ↑ ICP
(+) ARDS: Target DP < 15 cmH2O [c]
(+) ARDS: Target P_{plat} < 30 cmH2O [c]

Fig. 2. Ventilatory targets in SABI. ARDS, acute respiratory distress syndrome; CBF, cerebral blood flow; CBV, cerebral blood volume; CPP, cerebral perfusion pressure; DP, driving pressure; HOB, head of bed; ICP, intracranial pressure; JVO, jugular venous outflow; LTVV, low tidal volume ventilation; MAP, mean arterial pressure; $Paco_2$, arterial carbon dioxide partial pressure; Pao_2, arterial oxygen partial pressure; $PbtO_2$, brain tissue oxygen pressure; PBW, per body weight; P_{Plat}, plateau pressure; PEEP, positive end-expiratory pressure; Vt, tidal volume. [a]Based on ESICM guidelines, [b]Based on BTF guidelines, limited evidence about impact on outcomes, and [c]Thresholds based on ARDS populations, not validated in SABI.

centrally mediated dyssynchronies via increased central stimulation.[37,38] The impact of ventilator mode on paroxysmal sympathetic hyperactivity warrants further investigation.

Arterial Oxygen and Carbon Dioxide Goals

The recent ESICM guidelines strongly recommend targets within generally standard normal physiologic ranges for carbon dioxide ($Paco_2$ 35–45 mm Hg), as well as slightly higher arterial oxygen partial pressure (Pao_2) goal of 80–120 mm Hg.[36] $Paco_2$ acts as a fundamental regulator of cerebral blood flow, and both hypercarbia and hypocarbia have been associated with worse clinical outcomes.[39–41] Hypercarbia causes acidosis and cerebral vasodilation, which can result in elevated ICP.[32,33] Hypocarbia causes alkalosis and cerebral vasoconstriction, which can result in cerebral ischemia. Therapeutic hyperventilation can be used as a temporizing measure for reducing acutely elevated ICP but in general is only thought to be effective short-term (for minutes-hours), with risks of cerebral ischemia while hypocapnic (within hours) and rebound ICP spikes on normalization.[30,36] As part of the tiered approach to treat elevated ICP in TBI, slightly lower ($Paco_2$ 30–35 mm Hg) therapeutic ranges may be prudent in select cases.[42] In contrast, mild hypercapnia may be beneficial in optimizing cerebral blood flow to match cerebral oxygen delivery needs. The recent TAME trial sought to use the cerebral vasodilatory effect of hypercarbia to facilitate improved perfusion following cardiac arrest but found no significant difference in outcomes for patients that were hypercapnic ($Paco_2$ 50–55 mm Hg) compared with the normocapnic group ($Paco_2$ 35–45 mm Hg).[43]

Although exact thresholds for Pao_2 remain to be established, both hyperoxia and hypoxia have been associated with worse outcome in SABI, following a U-shaped curve.[28–30,44–46] The ESICM guidelines recommended a higher range than typical Pao_2 goals, with the rationale that the acutely injured, hypermetabolic brain may be more prone to hypoxemia. The value of a brain tissue oxygen pressure ($PbtO_2$)-guided approach is currently under investigation. For this treatment algorithm, a $PbtO_2$ probe is inserted in conjunction with an ICP monitor to concurrently monitor these 2 values. $PbtO_2$ is thought to reflect cerebral perfusion and the diffusion of oxygen across the

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

BBB. Several studies in TBI and SAH have shown associations between mortality and $PbtO_2$ levels, and a phase 2 randomized control trial (RCT) suggested outcome benefits with $PbtO_2$-directed therapy.[29,47–52] The BOOST-3 trial is further assessing the potential of ICP plus $PbtO_2$-guided management to improve the Glasgow Outcome Scale at 6 months.[53] The recently published OXY-TC trial did not demonstrate a reduction of poor neurologic outcomes at 6-month with ICP and $PbtO_2$-guided treatment versus ICP monitoring alone.[54]

Tidal Volume

In the landmark ARMA trial, patients receiving tidal volumes (Vt) of 4 to 6 cc/kg of predicted body weight (PBW) as compared with 12 cc/kg PBW demonstrated an 8.8% absolute reduction in mortality and fewer MV days.[55] Since then, low tidal volume ventilation (LTVV) is considered standard of care in ARDS, and benefits have also been shown in non-ARDS populations.[55–60] However, patients with elevated ICP were excluded from ARMA. In the ARMA trial, a statistically significant increase in mean P_aCO_2 between the 4 to 6 cc/kg group and 12 cc/kg group was noted. This was thought to be an acceptable trade-off given the improvements in clinical outcomes, leading to the paradigm of permissive hypercapnia in ARDS management. However, higher P_aCO_2 may exacerbate ICP crises in SABI, which may shift the trade off toward potential harm from permissive hypercapnia.

Insufficient knowledge about risks and benefits of LTVV in SABI may result in detrimental outcomes such as herniation in subgroups of patients at risk for elevated ICP; however, these concerns about using LTVV may result in clinicians withholding a therapy with proven benefits. For example, in the recent VENTILO survey, only about half of clinicians were comfortable using 4 to 6 cc/kg PBW in patients with TBI with moderate-to-severe respiratory hypoxemic respiratory failure.[61] However, a secondary analysis of 3 cohort studies showed increasing LTVV utilization between 2004 and 2016 and higher proportion of days spent receiving less than 8 mL/kg PBW. Other studies have also shown that the use of high Vt (>9 mL/kg PBW) is associated with the development of ARDS in various SABI subtypes.[62]

The ESICM guidelines strongly recommend LTVV in brain-injured patients with ARDS without elevated ICP but were unable to make concrete recommendations in patients with ARDS and elevated ICP.[36] In this population, risks and benefits must be weighed carefully based on oxygenation, lung compliance, and neurologic examination plus imaging. Vigilant monitoring of $Paco_2$ is warranted, and an invasive ICP monitor may be considered in high-risk patients to determine individualized $Paco_2$ thresholds and guide use of ICP-lowering therapies. Moreover, a recent small randomized controlled trial suggested that initial ICP, cerebral compliance, and mechanical power (MP) can be predictive of which patients develop elevated ICP with LTVV.[63] Strategies to increase respiratory rate and augment minute ventilation to avoid $Paco_2$ elevations are controversial and warrant further exploration.

Positive End-Expiratory Pressure

Optimal positive end-expiratory pressure (PEEP) titration can promote alveolar expansion, optimize lung recruitment,[64] and enable more homogeneous distribution of MV forces.[65] Several RCTs have showed improved oxygenation with higher PEEP strategies,[66–68] and a meta-analysis of 3 RCTs showed a 5% absolute mortality benefit. However, excessive PEEP can result in impeded venous return due to increased intrathoracic and right atrial pressures, which may subsequently cause elevated ICP by impeding cerebral venous drainage. Higher PEEP may also decrease CPP in patients with impaired cerebral autoregulation, in part due to effects on preload.[7,8,63,69]

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

Studies investigating the impact of PEEP on ICP in SABI have been smaller single-center studies (<20 patients), included a mix of heterogeneous SABI subtypes with distinct pathophysiology, typically did not assess PEEP greater than 12 to 15 cm H_2O, showed mixed results regarding the impact of PEEP on ICP and CPP, and generally did not assess long-term neurologic outcomes.[63,70–79]

Some reports have suggested that the main mechanism resulting in CPP reductions seems to be a PEEP-dependent decrease in mean arterial pressure (MAP), with ICP and CPP improving once MAP is restored.[74,76] One study found that the impact of PEEP on ICP was substantially higher in patients with alveolar hyperinflation based on static volume–pressure curves.[78] PEEP-mediated ICP elevations have also been shown to occur preferentially in patients with decreased respiratory compliance.[61,70,80]

To date, there is insufficient data to predict the impact of PEEP titration on ICP and ultimately, neurologic outcome. What is becoming more recognized is tolerability of higher PEEP in a subpopulation with intact cerebral autoregulation and preserved lung compliance. PEEP titration in patients with SABI should be deliberate and carefully monitored, with special attention toward maintaining MAP and avoiding alveolar hyperdistention. Invasive and noninvasive techniques to monitor ICP and cerebral perfusion should be considered in patients at high risk for herniation, especially at higher PEEP (>12 cm H_2O). The simultaneous use of lung and brain ultrasound to guide PEEP titration in SABI has been described.[73]

Plateau Pressure and Driving Pressure

The ARMA trial demonstrated lower mortality and fewer MV days in patients with plateau pressure (P_{Plat}) of 25 to 30 cm H_2O, compared with those receiving P_{Plat} 45 to 50 cm H_2O.[55] The optimal P_{Plat} for patients with SABI remains unknown. A special consideration, again, is the potential risk of elevated ICP when adjusting Vt or titrating PEEP to reduce P_{Plat}. In addition, high P_{plat} may result in ICP elevations due to decreased cerebral venous return. Until further evidence is available, it is reasonable to avoid P_{Plat} greater than 30 cm H_2O to reduce the risk of barotrauma and ventilator-induced lung injury (VILI). Patients with concomitant ARDS and elevated ICP should be managed with an individualized strategy, with gradual adjustments of Vt and PEEP while observing ICP effects and treating elevated ICP as needed. It is also important to recognize and address other potential contributors to elevated P_{Plat} independent of Vt, including alveolar overdistention, pulmonary congestion, pneumothorax, or migration of the endotracheal tube.

Driving pressure (ΔP), measured as the difference between P_{plat} and PEEP, was the ventilatory variable most strongly associated with mortality in a secondary analysis of 9 RCTs, independent of Vt.[81] This association has been confirmed in other ARDS cohorts.[82–85] As such, some have proposed that ΔP should be used as a physiologic target in patients with ARDS.[86] In SABI, an observational study of 986 patients showed that higher ΔP was associated with an increased risk of ARDS, whereas this association was not seen with Vt and PEEP.[62] Moreover, a preplanned secondary analysis of the TTM2-trial demonstrated associations between ΔP and mortality as well as neurologic outcome at 6 months in patients with postanoxic encephalopathy.[87] More data from RCTs directly examining the effects of ΔP is needed to establish as causal relationship. Clinically, ΔP can be modified by gradually lowering Vt and adjusting PEEP to optimize alveolar recruitment, while considering the impact on ICP.

Mechanical Power

The concept of MP as a determinant of VILI has gained increasing attention. MP is defined as the total energy transferred by the ventilator to the respiratory system

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

per each respiratory cycle, which is derived from several ventilator variables including pressure, volume, flow, and respiratory rate.[87–89] MP has been associated with mortality in ARDS populations,[90,91] and recently in several SABI cohorts.[92,93] A retrospective single-center study including 529 patients with mixed SABI subtypes demonstrated an association between MP in the first 24 hours and ICU mortality, with MP being a stronger predictor of mortality than GCS.[92] A secondary analysis of the TTM2-trial demonstrated associations between MP and mortality but not neurologic outcomes.[94] Another recent secondary analysis of a prospective, observational multicenter study including 1512 patients with SABI showed associations between MP at hospital day 1, 3, and 7 with mortality, reintubation, and development of ARDS.[93]

A causal relationship between MP and outcomes remains to be further evaluated in RCTs, and ventilatory strategies to limit MP should be viewed as exploratory. Further important areas of research include establishing critical MP thresholds in SABI versus other ICU populations, comparing the impact of MP in different SABI subtypes with distinct pathophysiology, and accounting for interactions between MP and ICP.

RESCUE MANEUVERS

Prone Positioning

The PROSEVA trial demonstrated a 17% absolute mortality reduction in patients with a ratio of partial pressure of arterial oxygen to the fraction of inspired oxygen (P/F ratio) less than 150 who were prone for greater than 16 hours per day[95]; however, patients with ICP greater than 30 were excluded. There are several important considerations and logistical challenges when proning patients with SABI, such as cervical spine instability in trauma, positioning in patients with cranial bone flaps, dislodgment of ICP monitors, or inadequate cerebrospinal fluid (CSF) flow from external ventricular drain (EVDs). A main concern is also the potential for ICP elevations due to increased abdominal pressure, decreased head elevation, and compression of neck veins affecting cerebral venous drainage. Studies investigating effects of prone positioning on ICP are all limited to date due to small sample size, mixed subtypes of brain injuries, various degrees of head elevation, and short proning durations (which was a key criticism of negative proning trials before PROSEVA).[96–99] A recent systematic review summarizing these studies showed that many demonstrated statistically significant transient elevations in ICP by 5 to 15 mm Hg, with mixed effects on CPP, while also showing substantial improvement of Pa_{O_2} and $PbtO_2$.[96] The clinical relevance of these findings is not clear because long-term outcomes are not reported. Given the substantial mortality benefit in ARDS and potential impacts on cerebral oxygen delivery, proning should be strongly considered in patients with SABI and concomitant moderate-to-severe ARDS. In cases with concerns about ICP, invasive monitoring can be used to guide adjunct ICP treatments such as hyperosmolar therapy or CSF diversion. Other measures to mitigate ICP elevations include aggressive bowel regimens, padding to decrease abdominal pressure, and modifying the prone angle to improve cerebral venous return. In addition, CPP can be supported via MAP augmentation with vasopressors. In cases where proning is thought to be contraindicated, supine chest wall compression with the use of anterior chest weights can be considered.

Venovenous Extracorporeal Membrane Oxygenation

Venovenous extracorporeal membrane oxygenation (VV-ECMO) can be an effective rescue therapy in severe refractory ARDS. The CESAR trial demonstrated lower mortality in patients with ARDS who were transferred to an extracorporeal membrane oxygenation (ECMO) center,[100] and the EOLIA trial showed a nonsignificant trend

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

toward improved outcome with VV-ECMO.[6] Historically, SABI had been considered a relative contraindication for ECMO due to concerns about hematoma expansion or hemorrhagic conversion with the use of therapeutic anticoagulation, decreased cerebral venous return with large venous cannulas placed in the internal jugular vein, and hesitations around resource utilization in patients with anticipated poor neurologic outcomes. However, there have been significant advances in the past years and increasing utilization of ECMO in SABI.[101] Heparin-bonded circuits and polymethyl pentene oxygenators have allowed for extended VV-ECMO support with low-dose or no systemic anticoagulation without premature oxygenator failure or excessive thrombotic complications.[101,102] Femoral-femoral cannula configurations avoid impairments in cerebral venous drainage. In general, rapid $Paco_2$ correction or extreme fluctuations in $Paco_2$ should be avoided by using low initial sweep gas rates and slow titration in general to minimize ischemic or hemorrhagic complications.[103] Overall, ECMO is feasible in highly selected patients with SABI and should be considered on a case-by-case basis with nuanced consideration of anticipated neurologic outcome.

DYSSYNCHRONY AND BREATHING PATTERNS

The full examination of management of VD and interpretation of ventilator waveforms to assist with such is beyond the scope of this article; however, we will highlight some key considerations specific to SABI. VD develops when there is a mismatch between patient and ventilator inspiratory and expiratory times. Respiratory drive and rhythm are controlled via the brainstem with cortical input, thus patients with SABI are at an increased risk for VD. This can develop due to issues with triggering breaths, oxygen flow, and improper cycling. Various types of centrally mediated VD are depicted in **Table 1**. Separately, agitation and patient discomfort may also lead to VD.

VD can result in inadequate oxygenation and ventilation, increased work of breathing, agitation, and patient discomfort, leading to increasing sedation requirements, which may result in increased MV days. VD can also impede venous return via increased intrathoracic pressure, which can lead to increased ICP, decreased brain perfusion and brain tissue oxygenation. In such severe clinically significant cases of VD, neuromuscular blockade (NMB) can be used to effectively eliminate any patient-initiated efforts. This, however, limits the ability for clinicians to track the patient's neurologic status. Another strategy is to blunt the respiratory drive using sedative medications.[104] With both NMB and sedation, careful titration of ventilator settings will need to be used to avoid inadvertent hypercapnia from decreased respiratory drive. With heavy sedation, patients may experience "paradoxic VD" termed reverse triggering, where the ventilator delivered breath triggers diaphragmatic effort.[105] Depending on the timing of this diaphragmatic effort, this may result in additional tidal volume being delivered or triggering a second breath. In patients with impaired intracranial compliance, this may lead to impaired venous return and increased ICP, which can counteract decreased cerebral metabolic demand from sedation.

In patients with SABI, often the need for MV may continue despite resolution of acute lung injury due to impaired airway protection. For such patients, spontaneous ventilator modes may minimize the development of VD or improve VD compared with a controlled mode, without requiring heavy sedation. In patients with a Cheyne-Stokes breathing pattern, this may result in triggering a backup control mode ventilation particularly during prolonged apneic pauses or periods of lower respiratory effort. This can be modulated by extending the apnea alarm times or decreasing the pressure or flow trigger sensitivities. In centrally mediated tachypnea,

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

ARTICLE IN PRESS

Table 1
Centrally mediated breathing patterns and neuroanatomical correlates

Breathing Patterns	Definition	Neuroanatomy	Prognosis	Wave form	Anatomy
Cheyne-Stokes	Oscillation between hyperventilation and hypoventilation	Bilateral or diencephalic insult typically but may occur anywhere between forebrain and pons	Typically portends good prognosis in stable patient however new onset could indicate impending herniation		
Short cycle periodic breathing	Faster Cheyne-Stokes pattern w/1–2 waxing breaths followed by 3–4 rapid breaths	Increased ICP, expanding posterior fossa lesion or lower pontine lesion			

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

Central neurogenic hyperventilation	RR 40–70 bpm; Po_2 >80 mm Hg and $Paco_2$ <40 mm Hg	Damage to central tegmentum of pons, ventral to aqueduct or fourth ventricle			
Apneustic breathing	Prolonged inspiratory gasp w/pause at end of respiration followed by expiration	Lesion of the dorsolateral lower half of the pons			

(*continued on next page*)

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

Table 1
(*continued*)

Breathing Patterns	Definition	Neuroanatomy	Prognosis	Wave form	Anatomy
Cluster breathing	Periodic breathing with irregular frequency and amplitude, along w/variable pauses between clusters of breaths	High medullary damage			
Ataxic breathing	Irregular in both rates and rhythm	Medullary lesions	Preterminal		

Abbreviations: bpm, breaths per minute, ICP, intracranial pressure.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

pressure support ventilation may be helpful in minimizing inadvertent hypocapnia from hyperventilation with static tidal volumes.

OTHER AREAS OF BRAIN–LUNG CONFLICT

Hemodynamic and Fluid Strategy

The FACTT trial demonstrated that conservative fluid management in ARDS decreased MV duration and ICU stay; however, there was no improvement in overall mortality.[106] In general, volume overload is strictly avoided in patients with ARDS,[107] and several studies have found reduced mortality with early diuresis.[108–110] In SABI, the mantra "dry lungs are happy lungs" and aggressive diuresis may pose a challenge in patients with acutely impaired brain perfusion. In particular, patients with acute proximal occlusions of the cerebral or cervical vasculature, those with a large ischemic penumbra, and those with SAH in cerebral vasospasm are at high risk for secondary brain injury due to cerebral ischemia.

Hypotension is associated with higher mortality and worse outcomes in all SABI subtypes.[28,111–113] Studies about the optimal volume status are often limited by crude modalities of volume status assessment such as total body balance. Both hypovolemia and hypervolemia in TBI and SAH have been associated with mortality.[114–117] Hyperosmolar therapies to treat elevated ICP can also affect volume and respiratory status. Mannitol causes an osmotic diuresis and may lead to decreased preload and cardiac output but can also exacerbate acute kidney injury. Hypertonic saline may increase preload and worsen pulmonary edema. It should be noted that both cause a transient shift of fluids to the intravascular compartment, which may affect cardiopulmonary physiology. Overall, volume management of patients with concurrent ARDS and SABI requires a tailored approach, using daily multimodal volume status assessments during critical time windows (eg, vasospasm period or while preserving an ischemic penumbra), and frequent reassessment of blood pressure and fluid targets. Additional imaging modalities such as CT-angiogram, CT-perfusion, or transcranial Dopplers may prove useful in stratifying the risk of cerebral ischemia, assessing cerebrovascular flow dynamics, and guiding cerebral physiology-based targets.

Steroids

After several negative trials of steroids in the treatment of ARDS, DEXA-ARDS was the first major RCT showing lower mortality and reduced MV days in patients with moderate-to-severe ARDS,[118] and multiple studies followed in COVID-ARDS populations demonstrating outcome benefits of steroids.[118–121] The use of steroids in SABI is controversial. Corticosteroids can reduce cerebral vasogenic edema by decreasing BBB permeability[122] and have shown benefits in subtypes of bacterial meningitis or brain tumors. However, studies in AIS,[123] SAH,[124,125] and ICH[126] did not show beneficial effects on outcomes but increased medical complications have been reported.[123–128] Corticosteroids in the postcardiac arrest population have been shown to improve return of spontaneous circulation rates when simultaneously infused with vasopressors but no improvement in neurologic outcomes has been shown.[129–131] In TBI, high-dose corticosteroids are contraindicated (Brain Trauma Foundation guidelines, level 1 recommendation)[42] based on the MRC CRASH trial, which demonstrated higher mortality at 2 weeks[128] and 6 months[127] with use of high-dose methylprednisolone.[127,128] With insufficient data to guide decisions about steroids in concomitant ARDS and SABI, more research is needed to establish which ARDS and SABI subpopulations may benefit or be harmed by steroids. Meanwhile, decisions

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

about steroids in this population need to be made on a case-by case basis; generally, they would be avoided in TBI, and other indications and contraindications for steroids in critically ill patients should be considered.

EXTUBATION DECISIONS AND TRACHEOSTOMY

The optimal strategy to liberate patients with SABI from MV is not known. The unique challenge in SABI is how to determine "extubation readiness" in patients with decreased level of consciousness and potentially impaired airway protective reflexes.

In several recent SABI extubation cohorts, rates of extubation failure within 2 to 7 days ranged between 15% and 40%; factors such as younger age, type of brain injury, higher GCS, intact gag or cough reflex, deglutition, prophylactic physical therapy, and lower frequency of suctioning were associated with successful extubation.[132–138] The role of GCS has been controversial, with mixed findings across studies. In the largest study to date, a prospective, observational multicenter trial including 1512 across 73 ICUs (ENIO), 19.4% of patients required reintubation within 5 days, and extubation failure rates ranged from 0% to 28.6% between countries. A predictive model with 20 variables predictors was derived from this cohort; however, its area under the curve (AUC) and predictive power were low.[138] A secondary analysis of ENIO found that extubation to noninvasive devices did not increase extubation success.[135] A prospective study in brain-injured patients with decreased levels of consciousness who met "extubation readiness" otherwise showed higher rates of pneumonia, MV, ICU length of stay, and cost of care in those who remained intubated for greater than 48 hours, suggesting that "waiting" is not necessarily beneficial.[139] However, it is always important to optimize quickly reversible factors, such as elevated ICP or seizures.

The question about ideal timing of tracheostomy also remains controversial but highly relevant. In ENIO, 21% of patients received a tracheostomy without an extubation attempt. Rates for tracheostomy are generally higher in mechanically ventilated patients with SABI (15%–35%) compared with the general ICU populations (10%–15%).[2,140,141] The SET score and Trach score were developed to anticipate the need for tracheostomy in patients[142,143] and to account for relevant factors such as subcortical or infratentorial location and SABI size, hydrocephalus, and midline shift. A meta-analysis of 10 studies including 503 patients with stroke and TBI found lower ICU length of stay and lower long-term mortality in patients with early tracheostomy (defined as <7 days) but the mortality benefit was no longer observed when excluding one study with high risk of bias.[134] A recent multicenter RCT in German and US Centers assessing early (within 3–5 days) versus late (10–14 days) tracheostomy in patients with stroke (AIS, ICH, and SAH) did not find any differences in long-term neurologic outcomes, mortality, ICU length of stay, sedation, or MV duration; however, 22% of patients who were randomized to the late tracheostomy group did not require a tracheostomy.[144] Benefits of early tracheostomy are insufficiently explored in the TBI population, where stimulation by the endotracheal tube may precipitate or exacerbate paroxysmal sympathetic hyperactivity or contribute to ICP crises. A prospective observational study from the CENTER-TBI cohort showed tracheostomy rates between 8% and 50% between countries and early tracheostomy rates between 0% and 17%, reflecting the variations in practice and lack of definite knowledge on this topic.[144]

Finally, information on long-term outcomes after tracheostomy in SABI is valuable to guide goals of care discussions. Current studies have shown that up to 80% of survivors with SABI who undergo tracheostomy placement are eventually decannulated[145];

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

however, mortality is as high as 30% to 40% at 1 year, and 30% to 50% are in a functionally dependent state at 6 to 12 months.[144,145] Neurologic disability, mortality, and decannulation rates vary based on type of brain injury, with SAH and TBI generally showing more favorable outcomes compared with AIS, ICH, and cardiac arrest.[146,147] More data are needed to further inform neuroprognostication and nuanced decisions about resuming life-sustaining treatments.[148]

SUMMARY

Acute respiratory failure is common in SABI, yet there is insufficient evidence to guide MV strategies as patients with SABI have been underrepresented in landmark studies. The relationship between brain–lung pathophysiology is complex and bidirectional, and at times management strategies between the lung and brain may conflict. Potential conflicts include utilization of LTVV, PEEP titration, P_{Plat} and ΔP optimization, and prone positioning in patients at risk for elevated ICP. Additional challenges include balancing the needs for sedation to ensure ventilator tolerance/synchrony with the importance of preserving the neurologic examination, volume status management in patients who may benefit from diuresis from a respiratory standpoint but are at risk for impaired cerebral perfusion, and treatment with corticosteroids, with some benefits shown in ARDS subpopulations but increased mortality in TBI. These concerns may be mitigated by close invasive and noninvasive neuromonitoring and require considerations of risks and benefits in the individual patient. More evidence is needed to guide optimal strategies in various SABI subpopulations. Our ability to predict successful extubation in this population remains limited but recent larger cohort studies have identified associations to inform clinical decisions. A recent RCT did not show benefits of early tracheostomy in stroke, and more data are needed in TBI. An important but understudied area is the interplay between extubation/tracheostomy decisions and neurologic prognostication.

DISCLOSURE

The authors declare no conflicts of interest or financial disclosures relevant to this article.

REFERENCES

1. Pham T, Heunks L, Bellani G, et al. Weaning from mechanical ventilation in intensive care units across 50 countries (WEAN SAFE): a multicentre, prospective, observational cohort study. Lancet Respir Med 2023;11(5):465–76.
2. Pelosi P, Ferguson ND, Frutos-Vivar F, et al. Management and outcome of mechanically ventilated neurologic patients. Crit Care Med 2011;39(6):1482–92.
3. Papazian L, Forel J-M, Gacouin A, et al. Neuromuscular blockers in early acute respiratory distress syndrome. N Engl J Med 2010;363(12):1107–16.
4. National Heart Lung, Blood Institute PETAL Clinical Trials Network, Moss M, Huang DT, et al. Early neuromuscular blockade in the acute respiratory distress syndrome. N Engl J Med 2019;380(21):1997–2008.
5. Peek GJ, Clemens F, Elbourne D, et al. CESAR: conventional ventilatory support vs extracorporeal membrane oxygenation for severe adult respiratory failure. BMC Health Serv Res 2006;6:163.
6. Combes A, Hajage D, Capellier G, et al. Extracorporeal membrane oxygenation for severe acute respiratory distress syndrome. N Engl J Med 2018;378(21):1965–75.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

7. Matin N, Sarhadi K, Crooks CP, et al. Brain-lung crosstalk: management of concomitant severe acute brain injury and acute respiratory distress syndrome. Curr Treat Options Neurol 2022;24(9):383–408.
8. Mrozek S, Constantin J-M, Geeraerts T. Brain-lung crosstalk: implications for neurocritical care patients. World J Crit Care Med 2015;4(3):163–78.
9. Aisiku IP, Yamal J-M, Doshi P, et al. The incidence of ARDS and associated mortality in severe TBI using the Berlin definition. J Trauma Acute Care Surg 2016; 80(2):308–12.
10. Pitoni S, D'Arrigo S, Grieco DL, et al. Tidal volume lowering by instrumental dead space reduction in brain-injured ARDS patients: effects on respiratory mechanics, gas exchange, and cerebral hemodynamics. Neurocritical Care 2021;34(1):21–30.
11. Johnson NJ, Caldwell E, Carlbom DJ, et al. The acute respiratory distress syndrome after out-of-hospital cardiac arrest: incidence, risk factors, and outcomes. Resuscitation 2019;135:37–44.
12. Zhao J, Xuan N-X, Cui W, et al. Neurogenic pulmonary edema following acute stroke: the progress and perspective. Biomed Pharmacother 2020;130:110478.
13. Rajajee V, Riggs B, Seder DB. Emergency neurological life support: airway, ventilation, and sedation. Neurocritical Care 2017;27(Suppl 1):4–28.
14. Pitts T. Airway protective mechanisms. Lung 2014;192(1):27–31.
15. Taran S, Wahlster S, Robba C. Ventilatory targets following brain injury. Curr Opin Crit Care 2023;29(2):41–9.
16. Holland MC, Mackersie RC, Morabito D, et al. The development of acute lung injury is associated with worse neurologic outcome in patients with severe traumatic brain injury. J Trauma 2003;55(1):106–11.
17. Rincon F, Ghosh S, Dey S, et al. Impact of acute lung injury and acute respiratory distress syndrome after traumatic brain injury in the United States. Neurosurgery 2012;71(4):795–803.
18. Kahn JM, Caldwell EC, Deem S, et al. Acute lung injury in patients with subarachnoid hemorrhage: incidence, risk factors, and outcome. Crit Care Med 2006;34(1):196–202.
19. Solenski NJ, Haley EC, Kassell NF, et al. Medical complications of aneurysmal subarachnoid hemorrhage: a report of the multicenter, cooperative aneurysm study. Participants of the Multicenter Cooperative Aneurysm Study. Crit Care Med 1995;23(6):1007–17.
20. Gruber A, Reinprecht A, Illievich UM, et al. Extracerebral organ dysfunction and neurologic outcome after aneurysmal subarachnoid hemorrhage. Crit Care Med 1999;27(3):505–14.
21. Elmer J, Hou P, Wilcox SR, et al. Acute respiratory distress syndrome after spontaneous intracerebral hemorrhage. Crit Care Med 2013;41(8):1992–2001.
22. Rincon F, Maltenfort M, Dey S, et al. The prevalence and impact of mortality of the acute respiratory distress syndrome on admissions of patients with ischemic stroke in the United States. J Intensive Care Med 2014;29(6):357–64.
23. Jakob DA, Lewis M, Benjamin ER, et al. Isolated traumatic brain injury: routine intubation for Glasgow Coma Scale 7 or 8 may be harmful. J Trauma Acute Care Surg 2021;90(5):874–9.
24. Irvin CB, Szpunar S, Cindrich LA, et al. Should trauma patients with a Glasgow Coma Scale score of 3 be intubated prior to hospital arrival? Prehospital Disaster Med 2010;25(6):541–6.
25. Gentleman D, Dearden M, Midgley S, et al. Guidelines for resuscitation and transfer of patients with serious head injury. BMJ 1993;307(6903):547–52.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

26. Hatchimonji JS, Dumas RP, Kaufman EJ, et al. Questioning dogma: does a GCS of 8 require intubation? Eur J Trauma Emerg Surg 2021;47(6):2073–9.
27. Doerfler S, Faerber J, McKhann GM, et al. The incidence and impact of secondary cerebral insults on outcome after aneurysmal subarachnoid hemorrhage. World Neurosurg 2018;114:e483–94.
28. Manley G, Knudson MM, Morabito D, et al. Hypotension, hypoxia, and head injury: frequency, duration, and consequences. Arch Surg 2001;136(10): 1118–23.
29. Lazaridis C, Ajith A, Mansour A, et al. Prediction of intracranial hypertension and brain tissue hypoxia utilizing high-resolution data from the BOOST-II clinical trial. Neurotrauma Reports 2022;3(1):473–8.
30. Siwicka-Gieroba D, Robba C, Gołacki J, et al. Cerebral oxygen delivery and consumption in brain-injured patients. J Personalized Med 2022;12(11).
31. Sharma D. Perioperative management of aneurysmal subarachnoid hemorrhage. Anesthesiology 2020;133(6):1283–305.
32. Roberts BW, Kilgannon JH, Chansky ME, et al. Association between postresuscitation partial pressure of arterial carbon dioxide and neurological outcome in patients with post-cardiac arrest syndrome. Circulation 2013;127(21):2107–13.
33. Roberts BW, Karagiannis P, Coletta M, et al. Effects of PaCO2 derangements on clinical outcomes after cerebral injury: a systematic review. Resuscitation 2015; 91:32–41.
34. Miller JD. Early insults to the injured brain. JAMA, J Am Med Assoc 1978;240(5): 439–42.
35. Robinson N. In patients with head injury undergoing rapid sequence intubation, does pretreatment with intravenous lignocaine/lidocaine lead to an improved neurological outcome? A review of the literature. Emerg Med J 2001;18(6): 453–7.
36. Robba C, Poole D, McNett M, et al. Mechanical ventilation in patients with acute brain injury: recommendations of the European Society of Intensive Care Medicine consensus. Intensive Care Med 2020;46(12):2397–410.
37. Luo X-Y, He X, Zhou Y-M, et al. Patient-ventilator asynchrony in acute brain-injured patients: a prospective observational study. Ann Intensive Care 2020; 10(1):144.
38. Epstein SK. How often does patient-ventilator asynchrony occur and what are the consequences? Respir Care 2011;56(1):25–38.
39. Davis DP, Meade W, Sise MJ, et al. Both hypoxemia and extreme hyperoxemia may be detrimental in patients with severe traumatic brain injury. J Neurotrauma 2009;26(12):2217–23.
40. Helmerhorst HJF, Schultz MJ, van der Voort PHJ, et al. Bench-to-bedside review: the effects of hyperoxia during critical illness. Crit Care 2015;19(1):284.
41. Alali AS, Temkin N, Vavilala MS, et al. Matching early arterial oxygenation to long-term outcome in severe traumatic brain injury: target values. J Neurosurg 2019;132(2):537–44.
42. Carney N, Totten AM, O'Reilly C, et al. Guidelines for the management of severe traumatic brain injury, fourth edition. Neurosurgery. 2017;80(1):6-15. doi:10.1227/NEU.0000000000001432.
43. Eastwood G, Nichol AD, Hodgson C, et al. Mild hypercapnia or normocapnia after out-of-hospital cardiac arrest. N Engl J Med 2023;389(1):45–57.
44. Menon DK, Coles JP, Gupta AK, et al. Diffusion limited oxygen delivery following head injury. Crit Care Med 2004;32(6):1384–90.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

45. Okonkwo DO, Shutter LA, Moore C, et al. Brain oxygen optimization in severe traumatic brain injury phase-II: a phase II randomized trial. Crit Care Med 2017;45(11):1907–14.
46. Sheinberg M, Kanter MJ, Robertson CS, et al. Continuous monitoring of jugular venous oxygen saturation in head-injured patients. J Neurosurg 1992;76(2):212–7.
47. Maloney-Wilensky E, Gracias V, Itkin A, et al. Brain tissue oxygen and outcome after severe traumatic brain injury: a systematic review. Crit Care Med 2009;37(6):2057–63.
48. Xie Q, Wu H-B, Yan Y-F, et al. Mortality and outcome comparison between brain tissue oxygen combined with intracranial pressure/cerebral perfusion pressure-guided therapy and intracranial pressure/cerebral perfusion pressure-guided therapy in traumatic brain injury: a meta-analysis. World Neurosurg 2017;100:118–27.
49. Narotam PK, Morrison JF, Nathoo N. Brain tissue oxygen monitoring in traumatic brain injury and major trauma: outcome analysis of a brain tissue oxygen-directed therapy. J Neurosurg 2009;111(4):672–82.
50. Gouvea Bogossian E, Diaferia D, Ndieugnou Djangang N, et al. Brain tissue oxygenation guided therapy and outcome in non-traumatic subarachnoid hemorrhage. Sci Rep 2021;11(1):16235.
51. Lubillo ST, Parrilla DM, Blanco J, et al. Prognostic value of changes in brain tissue oxygen pressure before and after decompressive craniectomy following severe traumatic brain injury. J Neurosurg 2018;128(5):1538–46.
52. Spiotta AM, Stiefel MF, Gracias VH, et al. Brain tissue oxygen-directed management and outcome in patients with severe traumatic brain injury. J Neurosurg 2010;113(3):571–80.
53. Bernard F, Barsan W, Diaz-Arrastia R, et al. Brain Oxygen Optimization in Severe Traumatic Brain Injury (BOOST-3): a multicentre, randomised, blinded-endpoint, comparative effectiveness study of brain tissue oxygen and intracranial pressure monitoring versus intracranial pressure alone. BMJ Open 2022;12(3):e060188.
54. Payen J-F, Launey Y, Chabanne R, et al. Intracranial pressure monitoring with and without brain tissue oxygen pressure monitoring for severe traumatic brain injury in France (OXY-TC): an open-label, randomised controlled superiority trial. Lancet Neurol 2023;22(11):1005–14.
55. Acute Respiratory Distress Syndrome Network, Brower RG, Matthay MA, et al. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med 2000;342(18):1301–8.
56. Futier E, Constantin J-M, Paugam-Burtz C, et al. A trial of intraoperative low-tidal-volume ventilation in abdominal surgery. N Engl J Med 2013;369(5):428–37.
57. Mascia L, Pasero D, Slutsky AS, et al. Effect of a lung protective strategy for organ donors on eligibility and availability of lungs for transplantation: a randomized controlled trial. JAMA 2010;304(23):2620–7.
58. Serpa Neto A, Cardoso SO, Manetta JA, et al. Association between use of lung-protective ventilation with lower tidal volumes and clinical outcomes among patients without acute respiratory distress syndrome: a meta-analysis. JAMA 2012;308(16):1651–9.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

59. Asehnoune K, Mrozek S, Perrigault PF, et al. A multi-faceted strategy to reduce ventilation-associated mortality in brain-injured patients. The BI-VILI project: a nationwide quality improvement project. Intensive Care Med 2017;43(7):957–70.
60. Roquilly A, Cinotti R, Jaber S, et al. Implementation of an evidence-based extubation readiness bundle in 499 brain-injured patients. a before-after evaluation of a quality improvement project. Am J Respir Crit Care Med 2013;188(8):958–66.
61. Picetti E, Pelosi P, Taccone FS, et al. VENTILatOry strategies in patients with severe traumatic brain injury: the VENTILO Survey of the European Society of Intensive Care Medicine (ESICM). Crit Care 2020;24(1):158.
62. Tejerina E, Pelosi P, Muriel A, et al. Association between ventilatory settings and development of acute respiratory distress syndrome in mechanically ventilated patients due to brain injury. J Crit Care 2017;38:341–5.
63. Beqiri E, Smielewski P, Guérin C, et al. Neurological and respiratory effects of lung protective ventilation in acute brain injury patients without lung injury: brain vent, a single centre randomized interventional study. Crit Care 2023;27(1):115.
64. Briel M, Meade M, Mercat A, et al. Higher vs lower positive end-expiratory pressure in patients with acute lung injury and acute respiratory distress syndrome: systematic review and meta-analysis. JAMA 2010;303(9):865–73.
65. Gattinoni L, Carlesso E, Brazzi L, et al. Positive end-expiratory pressure. Curr Opin Crit Care 2010;16(1):39–44.
66. Meade MO, Cook DJ, Guyatt GH, et al. Ventilation strategy using low tidal volumes, recruitment maneuvers, and high positive end-expiratory pressure for acute lung injury and acute respiratory distress syndrome: a randomized controlled trial. JAMA 2008;299(6):637–45.
67. Nishikimi M, Ohshimo S, Hamaguchi J, et al. High versus low positive end-expiratory pressure setting in patients receiving veno-venous extracorporeal membrane oxygenation support for severe acute respiratory distress syndrome: study protocol for the multicentre, randomised ExPress SAVER Trial. BMJ Open 2023;13(10):e072680.
68. Mercat A, Richard J-CM, Vielle B, et al. Positive end-expiratory pressure setting in adults with acute lung injury and acute respiratory distress syndrome: a randomized controlled trial. JAMA 2008;299(6):646–55.
69. Neto AS, Simonis FD, Barbas CSV, et al. Lung-protective ventilation with low tidal volumes and the occurrence of pulmonary complications in patients without acute respiratory distress syndrome: a systematic review and individual patient data analysis. Crit Care Med 2015;43(10):2155–63.
70. Boone MD, Jinadasa SP, Mueller A, et al. The effect of positive end-expiratory pressure on intracranial pressure and cerebral hemodynamics. Neurocritical Care 2017;26(2):174–81.
71. Videtta W, Villarejo F, Cohen M, et al. Effects of positive end-expiratory pressure on intracranial pressure and cerebral perfusion pressure. Acta Neurochir Suppl 2002;81:93–7.
72. Caricato A, Conti G, Della Corte F, et al. Effects of PEEP on the intracranial system of patients with head injury and subarachnoid hemorrhage: the role of respiratory system compliance. J Trauma 2005;58(3):571–6.
73. Robba C, Ball L, Battaglini D, et al. Effects of positive end-expiratory pressure on lung ultrasound patterns and their correlation with intracranial pressure in mechanically ventilated brain injured patients. Crit Care 2022;26(1):31.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

74. Georgiadis D, Schwarz S, Baumgartner RW, et al. Influence of positive end-expiratory pressure on intracranial pressure and cerebral perfusion pressure in patients with acute stroke. Stroke 2001;32(9):2088–92.
75. Huynh T, Messer M, Sing RF, et al. Positive end-expiratory pressure alters intracranial and cerebral perfusion pressure in severe traumatic brain injury. J Trauma 2002;53(3):488–92, discussion 492.
76. Muench E, Bauhuf C, Roth H, et al. Effects of positive end-expiratory pressure on regional cerebral blood flow, intracranial pressure, and brain tissue oxygenation. Crit Care Med 2005;33(10):2367–72.
77. McGuire G, Crossley D, Richards J, et al. Effects of varying levels of positive end-expiratory pressure on intracranial pressure and cerebral perfusion pressure. Crit Care Med 1997;25(6):1059–62.
78. Mascia L, Grasso S, Fiore T, et al. Cerebro-pulmonary interactions during the application of low levels of positive end-expiratory pressure. Intensive Care Med 2005;31(3):373–9.
79. Corradi F, Robba C, Tavazzi G, et al. Combined lung and brain ultrasonography for an individualized "brain-protective ventilation strategy" in neurocritical care patients with challenging ventilation needs. Crit Ultrasound J 2018;10(1):24.
80. Shapiro HM, Marshall LF. Intracranial pressure responses to PEEP in head-injured patients. J Trauma 1978;18(4):254–6.
81. Amato MBP, Meade MO, Slutsky AS, et al. Driving pressure and survival in the acute respiratory distress syndrome. N Engl J Med 2015;372(8):747–55.
82. Bellani G, Laffey JG, Pham T, et al. Epidemiology, patterns of care, and mortality for patients with acute respiratory distress syndrome in intensive care units in 50 countries. JAMA 2016;315(8):788–800.
83. Baedorf Kassis E, Loring SH, Talmor D. Mortality and pulmonary mechanics in relation to respiratory system and transpulmonary driving pressures in ARDS. Intensive Care Med 2016;42(8):1206–13.
84. Coppola S, Caccioppola A, Froio S, et al. Effect of mechanical power on intensive care mortality in ARDS patients. Crit Care 2020;24(1):246.
85. Aoyama H, Pettenuzzo T, Aoyama K, et al. Association of driving pressure with mortality among ventilated patients with acute respiratory distress syndrome: a systematic review and meta-analysis. Crit Care Med 2018;46(2):300–6.
86. Pelosi P, Ball L, Barbas CSV, et al. Personalized mechanical ventilation in acute respiratory distress syndrome. Crit Care 2021;25(1):250.
87. Robba C, Badenes R, Battaglini D, et al. Oxygen targets and 6-month outcome after out of hospital cardiac arrest: a pre-planned sub-analysis of the targeted hypothermia versus targeted normothermia after Out-of-Hospital Cardiac Arrest (TTM2) trial. Crit Care 2022;26(1):323.
88. Gattinoni L, Tonetti T, Cressoni M, et al. Ventilator-related causes of lung injury: the mechanical power. Intensive Care Med 2016;42(10):1567–75.
89. Cressoni M, Gotti M, Chiurazzi C, et al. Mechanical power and development of ventilator-induced lung injury. Anesthesiology 2016;124(5):1100–8.
90. Costa ELV, Slutsky AS, Brochard LJ, et al. Ventilatory variables and mechanical power in patients with acute respiratory distress syndrome. Am J Respir Crit Care Med 2021;204(3):303–11.
91. Serpa Neto A, Deliberato RO, Johnson AEW, et al. Mechanical power of ventilation is associated with mortality in critically ill patients: an analysis of patients in two observational cohorts. Intensive Care Med 2018;44(11):1914–22.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

92. Jiang X, Zhu Y, Zhen S, et al. Mechanical power of ventilation is associated with mortality in neurocritical patients: a cohort study. J Clin Monit Comput 2022; 36(6):1621–8.
93. Wahlster S, Sharma M, Taran S, et al. Utilization of mechanical power and associations with clinical outcomes in brain injured patients: a secondary analysis of the extubation strategies in neuro-intensive care unit patients and associations with outcome (ENIO) trial. Crit Care 2023;27(1):156.
94. Robba C, Badenes R, Battaglini D, et al. Ventilatory settings in the initial 72 h and their association with outcome in out-of-hospital cardiac arrest patients: a preplanned secondary analysis of the targeted hypothermia versus targeted normothermia after out-of-hospital cardiac arrest (TTM2) trial. Intensive Care Med 2022;48(8):1024–38.
95. Guérin C, Reignier J, Richard J-C, et al. Prone positioning in severe acute respiratory distress syndrome. N Engl J Med 2013;368(23):2159–68.
96. Wright JM, Gerges C, Shammassian B, et al. Prone position ventilation in neurologically ill patients: a systematic review and proposed protocol. Crit Care Med 2021;49(3):e269–78.
97. Reinprecht A, Greher M, Wolfsberger S, et al. Prone position in subarachnoid hemorrhage patients with acute respiratory distress syndrome: effects on cerebral tissue oxygenation and intracranial pressure. Crit Care Med 2003;31(6): 1831–8.
98. Roth C, Ferbert A, Deinsberger W, et al. Does prone positioning increase intracranial pressure? A retrospective analysis of patients with acute brain injury and acute respiratory failure. Neurocritical Care 2014;21(2):186–91.
99. Thelandersson A, Cider A, Nellgård B. Prone position in mechanically ventilated patients with reduced intracranial compliance. Acta Anaesthesiol Scand 2006; 50(8):937–41.
100. Peek GJ, Mugford M, Tiruvoipati R, et al. Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial. Lancet 2009;374(9698):1351–63.
101. Kurihara C, Walter JM, Karim A, et al. Feasibility of venovenous extracorporeal membrane oxygenation without systemic anticoagulation. Ann Thorac Surg 2020;110(4):1209–15.
102. Fina D, Matteucci M, Jiritano F, et al. Extracorporeal membrane oxygenation without therapeutic anticoagulation in adults: a systematic review of the current literature. Int J Artif Organs 2020;43(9):570–8.
103. Gajkowski EF, Herrera G, Hatton L, et al. ELSO guidelines for adult and pediatric extracorporeal membrane oxygenation circuits. ASAIO J 2022;68(2):133–52.
104. Mellott KG, Grap MJ, Munro CL, et al. Patient-ventilator dyssynchrony: clinical significance and implications for practice. Crit Care Nurse 2009;29(6):41–55, quiz 1 p following 55.
105. Antonogiannaki E-M, Georgopoulos D, Akoumianaki E. Patient-ventilator dyssynchrony. Korean J Crit Care Med 2017;32(4):307–22.
106. Silversides JA, McAuley DF, Blackwood B, et al. Fluid management and deresuscitation practices: a survey of critical care physicians. J Intensive Care Soc 2020;21(2):111–8.
107. National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network, Wiedemann HP, Wheeler AP, et al. Comparison of two fluid-management strategies in acute lung injury. N Engl J Med 2006; 354(24):2564–75.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

108. Seitz KP, Caldwell ES, Hough CL. Fluid management in ARDS: an evaluation of current practice and the association between early diuretic use and hospital mortality. J Intensive Care 2020;8:78.
109. Zhang R, Chen H, Gao Z, et al. The effect of loop diuretics on 28-day mortality in patients with acute respiratory distress syndrome. Front Med 2021;8:740675.
110. Griffiths MJD, McAuley DF, Perkins GD, et al. Guidelines on the management of acute respiratory distress syndrome. BMJ Open Respir Res 2019;6(1):e000420.
111. Stead LG, Gilmore RM, Decker WW, et al. Initial emergency department blood pressure as predictor of survival after acute ischemic stroke. Neurology 2005; 65(8):1179–83.
112. Vemmos KN, Tsivgoulis G, Spengos K, et al. U-shaped relationship between mortality and admission blood pressure in patients with acute stroke. J Intern Med 2004;255(2):257–65.
113. Wohlfahrt P, Krajcoviechova A, Jozifova M, et al. Low blood pressure during the acute period of ischemic stroke is associated with decreased survival. J Hypertens 2015;33(2):339–45.
114. Zhao Z, Wang D, Jia Y, et al. Analysis of the association of fluid balance and short-term outcome in traumatic brain injury. J Neurol Sci 2016;364:12–8.
115. Clifton GL, Miller ER, Choi SC, et al. Fluid thresholds and outcome from severe brain injury. Crit Care Med 2002;30(4):739–45.
116. Wijdicks EF, Vermeulen M, ten Haaf JA, et al. Volume depletion and natriuresis in patients with a ruptured intracranial aneurysm. Ann Neurol 1985;18(2):211–6.
117. Wiegers EJA, Lingsma HF, Huijben JA, et al. Fluid balance and outcome in critically ill patients with traumatic brain injury (CENTER-TBI and OzENTER-TBI): a prospective, multicentre, comparative effectiveness study. Lancet Neurol 2021; 20(8):627–38.
118. Villar J, Ferrando C, Martínez D, et al. Dexamethasone treatment for the acute respiratory distress syndrome: a multicentre, randomised controlled trial. Lancet Respir Med 2020;8(3):267–76.
119. Yoshihro S, Taito S, Yatabe T. The influence of steroid type on outcomes in patients with acute respiratory distress syndrome. J Intensive Care 2023;11(1):32.
120. Khilnani GC, Hadda V. Corticosteroids and ARDS: a review of treatment and prevention evidence. Lung India 2011;28(2):114–9.
121. Ma S, Xu C, Liu S, et al. Efficacy and safety of systematic corticosteroids among severe COVID-19 patients: a systematic review and meta-analysis of randomized controlled trials. Signal Transduct Targeted Ther 2021;6(1):83.
122. Witt KA, Sandoval KE. Steroids and the blood-brain barrier: therapeutic implications. Adv Pharmacol 2014;71:361–90.
123. Sandercock PA, Soane T. Corticosteroids for acute ischaemic stroke. Cochrane Database Syst Rev 2011;2011(9):CD000064.
124. Feigin VL, Anderson N, Rinkel GJE, et al. Corticosteroids for aneurysmal subarachnoid haemorrhage and primary intracerebral haemorrhage. Cochrane Database Syst Rev 2005;3:CD004583.
125. Mistry AM, Mistry EA, Ganesh Kumar N, et al. Corticosteroids in the management of hyponatremia, hypovolemia, and vasospasm in subarachnoid hemorrhage: a meta-analysis. Cerebrovasc Dis 2016;42(3–4):263–71.
126. Poungvarin N, Bhoopat W, Viriyavejakul A, et al. Effects of dexamethasone in primary supratentorial intracerebral hemorrhage. N Engl J Med 1987;316(20): 1229–33.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

127. Edwards P, Arango M, Balica L, et al. Final results of MRC CRASH, a randomised placebo-controlled trial of intravenous corticosteroid in adults with head injury-outcomes at 6 months. Lancet 2005;365(9475):1957–9.
128. Roberts I, Yates D, Sandercock P, et al. Effect of intravenous corticosteroids on death within 14 days in 10008 adults with clinically significant head injury (MRC CRASH trial): randomised placebo-controlled trial. Lancet 2004;364(9442):1321–8.
129. Mentzelopoulos SD, Malachias S, Chamos C, et al. Vasopressin, steroids, and epinephrine and neurologically favorable survival after in-hospital cardiac arrest: a randomized clinical trial. JAMA 2013;310(3):270–9.
130. Andersen LW, Granfeldt A. Vasopressin and methylprednisolone vs placebo and return of spontaneous circulation in patients with in-hospital cardiac arrest—reply. JAMA 2022;327(5):487.
131. Shah K, Mitra AR. Use of corticosteroids in cardiac arrest-A systematic review and meta-analysis. Crit Care Med 2021;49(6):e642–50.
132. Jaber S, Quintard H, Cinotti R, et al. Risk factors and outcomes for airway failure versus non-airway failure in the intensive care unit: a multicenter observational study of 1514 extubation procedures. Crit Care 2018;22(1):236.
133. Godet T, Chabanne R, Marin J, et al. Extubation failure in brain-injured patients: risk factors and development of a prediction score in a preliminary prospective cohort study. Anesthesiology 2017;126(1):104–14.
134. McCredie VA, Ferguson ND, Pinto RL, et al. Airway management strategies for brain-injured patients meeting standard criteria to consider extubation. A prospective cohort study. Ann Am Thorac Soc 2017;14(1):85–93.
135. Taran S, Diaz-Cruz C, Perrot B, et al. Association of non-invasive respiratory support with extubation outcomes in brain-injured patients receiving mechanical ventilation: a secondary analysis of ENIO. Am J Respir Crit Care Med 2023. https://doi.org/10.1164/rccm.202212-2249OC.
136. Anderson CD, Bartscher JF, Scripko PD, et al. Neurologic examination and extubation outcome in the neurocritical care unit. Neurocritical Care 2011;15(3):490–7.
137. Castro AAM, Cortopassi F, Sabbag R, et al. Respiratory muscle assessment in predicting extubation outcome in patients with stroke. Arch Bronconeumol 2012;48(8):274–9.
138. Cinotti R, Pelosi P, Schultz MJ, et al. Extubation strategies in neuro-intensive care unit patients and associations with outcomes: the ENIO multicentre international observational study. Ann Transl Med 2020;8(7):503.
139. Coplin WM, Pierson DJ, Cooley KD, et al. Implications of extubation delay in brain-injured patients meeting standard weaning criteria. Am J Respir Crit Care Med 2000;161(5):1530–6.
140. Alsherbini K, Goyal N, Metter EJ, et al. Predictors for tracheostomy with external validation of the stroke-related early tracheostomy score (SETscore). Neurocritical Care 2019;30(1):185–92.
141. Kurtz P, Fitts V, Sumer Z, et al. How does care differ for neurological patients admitted to a neurocritical care unit versus a general ICU? Neurocritical Care 2011;15(3):477–80.
142. Schönenberger S, Al-Suwaidan F, Kieser M, et al. The SETscore to predict tracheostomy need in cerebrovascular neurocritical care patients. Neurocritical Care 2016;25(1):94–104.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.

143. Szeder V, Ortega-Gutierrez S, Ziai W, et al. The TRACH score: clinical and radiological predictors of tracheostomy in supratentorial spontaneous intracerebral hemorrhage. Neurocritical Care 2010;13(1):40–6.
144. Bösel J, Niesen W-D, Salih F, et al. Effect of early vs standard approach to tracheostomy on functional outcome at 6 months among patients with severe stroke receiving mechanical ventilation: the SETPOINT2 randomized clinical trial. JAMA 2022;327(19):1899–909.
145. Wahlster S, Sharma M, Chu F, et al. Outcomes after tracheostomy in patients with severe acute brain injury: a systematic review and meta-analysis. Neurocritical Care 2021;34(3):956–67.
146. Wabl R, Williamson CA, Pandey AS, et al. Long-term and delayed functional recovery in patients with severe cerebrovascular and traumatic brain injury requiring tracheostomy. J Neurosurg 2018;131(1):114–21.
147. Avesani R, Dambruoso F, Formisano R, et al. Considerations about status of severe acquired brain injuries in Italy. Int J Neurorehabilitation Eng 2017;04(02).
148. Lou W, Granstein JH, Wabl R, et al. Taking a chance to recover: families look back on the decision to pursue tracheostomy after severe acute brain injury. Neurocritical Care 2022;36(2):504–10.

Descargado para Anonymous User (n/a) en National Autonomous University of Mexico de ClinicalKey.es por Elsevier en febrero 07, 2024. Para uso personal exclusivamente. No se permiten otros usos sin autorización. Copyright ©2024. Elsevier Inc. Todos los derechos reservados.